INTRODUCTION

HISTORY OF LARYNGOSCOPY AND INTUBATION

In 1854, a Spanish vocal Pedagogist, Manuel Garcia (1805-1906) became the first man to visualize the functioning glottis in a human being.

On 23rd April 1895, Alfred Kirsten performed the first direct laryngoscopy.

In 1880 Scottish surgeon, William Macewen, (1848-1924) reported use of orotracheal intubation as an alternative to tracheotomy.

In 1913 Chevalier Jackson, reported high success in direct laryngoscopy.

Sir Robert Reynolds Macintosh (1897-1989), introduced his new curved laryngoscopy blade in 1943.

RESPONSE TO LARYNGOSCOPY

The induction of anaesthesia, laryngoscopy , tracheal intubation and surgical stimulation evoke cardiovascular responses leading to alteration in heart rate, cardiac rhythm and blood pressure. The response starts in 5 seconds , peaks within 1-2 minutes and returns to baseline in 5 minutes.

This sympatho adrenal response is of little significance in healthy patients but hazardous in patients with hypertension, coronary artery disease, cerebrovascular disease, intracranial pathology and hyperactive airways. In these patients the stress response should be attenuated.

Prof. King et al., in 1951 documented myocardial ischemic changes following laryngoscopy and intubation with increase in systolic blood pressure upto 40mmHg even in normotensive patients.

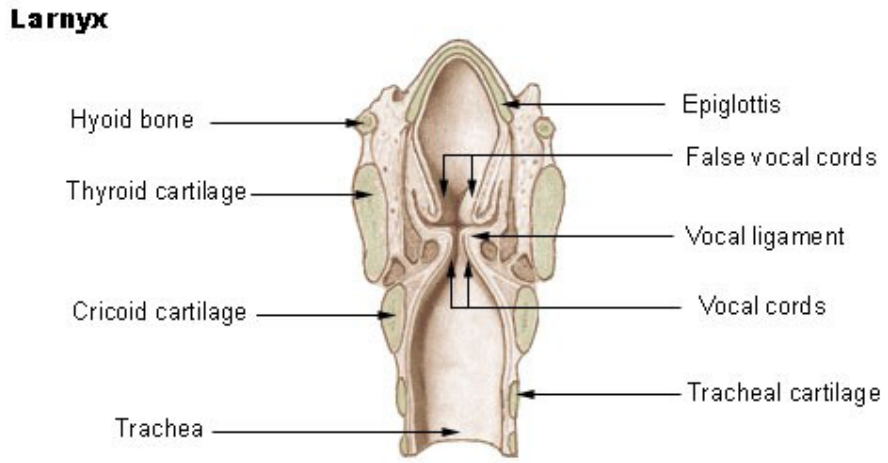
Various drugs are used to attenuate this stress response like local anaesthetics, narcotics, vasodilators, beta blockers, calcium channel blockers, Magnesium and centrally acting sympatholytics.

In the Institute of Anaesthesiology and critical care, Madras Medical College and Rajiv Gandhi Government General Hospital we have compared Esmolol and Magnesium Sulphate with placebo in attenuating hemodynamic stress response to laryngoscopy and intubation.

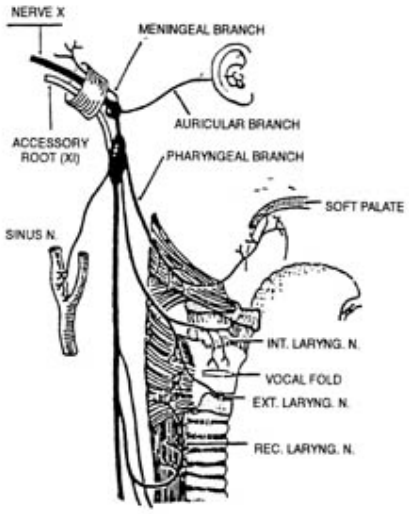
AIM OF THE STUDY

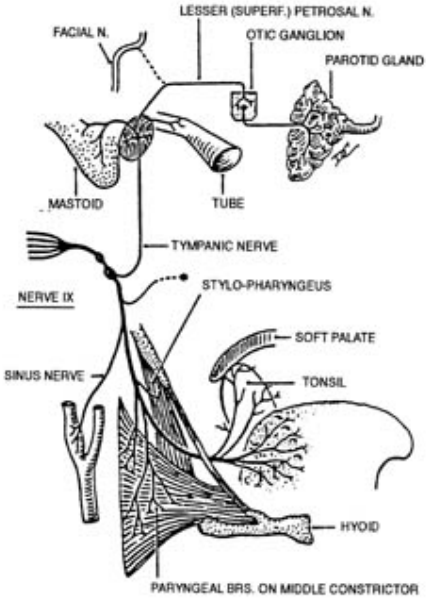
The haemodynamic stress response to laryngoscopy and intubation should be attenuated to balance myocardial oxygen supply and demand especially in coronary artery disease patients. It is also hazardous in patients with cerebrovascular disease, intracranial pathology and hyperactive airways. The aim of this study is to compare the effectiveness of Esmolol and Magnesium Sulphate for attenuation of haemodynamic stress response to laryngoscopy and intubation.

ANATOMY OF LARYNX



NERVE SUPPLY OF LARYNX





ANATOMY AND NERVE SUPPLY OF LARYNX

The pharynx is divided into nasopharynx, oropharynx and laryngopharynx. The epiglottis seperates the oropharynx from the laryngopharynx.

The palatine branch of the trigeminal nerve supplies the hard and soft palate. The anterior two third of the tongue is supplied by the lingual nerve and glossopharyngeal nerve supplies the posterior one third of the tongue. The glossopharyngeal nerve also supplies the roof of the pharynx.

The pharyngeal surface of the epiglottis is supplied by the glossopharyngeal nerve and the laryngeal surface is supplied by the vagus. The sensory supply of the supraglottic area is by the internal branch of superior laryngeal nerve.The motor branch of the recurrent laryngeal nerve supplies all the intrinsic muscles of the larynx except the cricothyroid, which is supplied by the external laryngeal nerve and the sensory branches of the recurrent laryngeal nerve supply the mucosa of the larynx below the vocal cords.

PHYSIOLOGY OF STRESS RESPONSE

The larynx is a heavily innervated sensory structure, so laryngoscopy and tracheal intubation stimulates these structures leading to the stress response. Hemodynamic stress response to laryngoscopy and intubation occurs as increase in heart rate and blood pressure due to reflex sympathetic discharge. The force and duration of laryngoscopy, hypoxia, hypercarbia, stimulation of carina by endotracheal tube , repeated and prolonged attempts affect the stress response.

This stress response starts within 5 seconds peaks within 1-2 minutes and returns to baseline in five minutes. This sympatho adrenal response is of little significance in healthy patients but hazardous in patients with hypertension, coronary artery disease , intra cranial pathology and hyperactive airways.

CARDIOVASCULAR RESPONSE:

Hypertension, tachycardia, bradycardia and dysrhythmias mediated by autonomic nervous system occurs. Hypertension, tachycardia , increase in cardiac work and oxygen consumption are mediated by sympathetic afferents via the cardioaccelerator fibres and sympathetic

chain ganglia. The polysynaptic pathway from 9th and 10th nerve afferents to sympathetic nervous system in the brain stem and spinal cord results in a diffuse autonomic response which includes widespread release of norepinephrine from the adrenergic terminals and release of epinephrine from the adrenal medulla.

One other reason for the hypertensive response is due to activation of renin- angiotensin system with release of renin from the renal juxtaglomerular apparatus, an end organ innervated by adrenergic nerve terminals.

Bradycardia caused by a rise in vagal tone in Sino atrial node is a monosynaptic reflex to a noxious stimuli.

RESPIRATORY SYSTEM:

1) Reflex glottis closure-laryngospasm

2) Decreases dead space

3) Increased airway resistance

4) Bronchospasm

STRESS RESPONSE IN PATHOLOGICAL STATES:

1) In patients with limited myocardial reserve , myocardial ischemia and failure can occur. It is essential to maintain the heart rate and blood pressure of the patient within 20% of the normal awake value. Heart rate should ideally be less than 110 beats per minute(ischemic threshold).

2) Intracranial vascular anomalies can rupture.

3) In patients with hyperactive airways bronchospasm and laryngospasm can occur.

4) Laryngoscopy and intubation can increase cerebral blood flow if autoregulation is impaired. The resultant increase in intracranial pressure can lead to brainstem herniation and death.

METHODS TO ATTENUATE INTUBATION RESPONSE:

1) Maintaining a deep plane of general anaesthesia using volatile anaesthetics. This dose of volatile anaesthetics required to block the cardiovascular responses to endotracheal intubation may result in profound cardiovascular depression. The volatile agents used are Halothane, Isoflurane and Sevoflurane.

2) Local anaesthetics . Lignocaine is used.

a) It is given as viscous gargle for oropharyngeal anaesthesia.

b) aerosol for intratracheal anaesthesia

c) intravenous

d) local instillation or topical spray over the vocal cords.

e)Regional nerve blocks

3) Vasodilators –Nitroglycerine

Sodium Nitroprusside

Hydralazine

4) Magnesium sulphate

5) Narcotics –Fentanyl, Sufentanil, Remifentanil, Morphine, Pethidine.

Fentanyl is the most commonly used narcotic.It is a potent analgesic, has a short duration of action, does not increase intracranial tension and has minimal circulatory changes.

6) Calcium channel blockers- Nifedipine, Nicardipine, Verapamil, Diltiazem

7) Adrenergic blockers –Beta blocker-Metoprolol, Esmolol

-Alpha blocker-Phentolamine

Alpha and beta blocker-Labetalol

8) Central sympatholytics-Clonidine and Dexmedetomidine. They act by decreasing central sympathetic outflow.

9)Sedatives and anxiolytics.

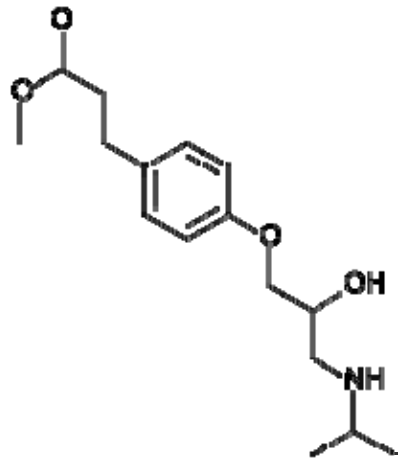
PHARMACOLOGY OF ESMOLOL

INTRODUCTION:

Esmolol is a beta adrenergic receptor antagonist. It binds to beta receptors and interferes with the ability of catecholamines to provoke a response.

STRUCTURE:

It is methyl p-(2-OH-3-isopropylaminopropoxy)hydrocinnamate hydrochloride.



The ester group in the para position accounts for high metabolic lability and short duration of action. This para substitution also confers cardioselectivity on Esmolol.

Mol.formula-C16H26NO4Cl

Molecular weight -331.8

MECHANISM OF ACTION:

Esmolol competitively inhibits beta adrenergic receptors. Beta receptors are G-protein coupled receptors and when stimulated activate adenylcyclase to produce cAMP.Net effect of beta stimulation is positive chronotropic, inotropic and dromotropic effect. Esmolol produces negative chronotropic, inotropic and dromotropic effect.

CLASSIFICATION:

It is a cardio selective beta 1 blocker.

PROPERTIES:

1) No significant intrinsic sympathomimetic activity

2) Membrane stabilizing effect at therapeutic doses

3) Cardioselective

4) 55% is protein bound

5) Clearance is by ester hydrolysis of the methyl side chain, so it has short duration of action

6) It has no active metabolites

7) Elimination half time is 9 minutes.

8) Distribution half-life is 2 minutes

9) It has rapid onset and short duration of action. Therapeutic effect occurs within 5 minutes that ceases within 10-30 minutes.

PHARMACOKINETICS:

Elimination half life is 9 minutes due to rapid hydrolysis by esterases in the cytosol of red blood cells. Elimination is independent of hepatic and renal function and plasma choline esterases. Distribution half life is 2 minutes. The major fraction of Esmolol is detectable in urine as acid metabolites which have longer elimination half life (4 hours) and slower clearance. However , the metabolites have only weak beta blocking actions which is about 1500 times less than the parent compound.

DOSE AND PREPARATION:

Loading dose is 0.5mg/kg over 60 seconds followed by infusion rate of 50-300 microgm/kg/min. Each ml contains 10mg Esmolol Hydrochloride , buffered with sodium acetate trihydrate and glacial acetic acid. Hydrochloric acid is added to adjust pH around 4.5-5.5

EFFECTS ON VARIOUS SYSTEMS:

1) CARDIO VASCULAR SYSTEM:

It decreases the heart rate, rate pressure product, cardiac index, systolic blood pressure and ejection fraction. It also decreases the rate of spontaneous phase 4 depolarisation. It has negative chronotropic, inotropic and dromotropic effects. It produces peripheral vasoconstriction due to unopposed alpha effect.

2) RESPIRATORY SYSTEM:

There is no bronchoconstriction since it is a cardioselective beta blocker.

3) METABOLIC EFECT:

It can lead to unrecognized hypoglycemia since tachycardia as a warning sign is blunted. It inhibits uptake of potassium into cells leading to hyperkalemia.

4) NERVOUS SYSTEM:

Fatigue and lethargy since it crosses the blood brain barrier.

5) FETUS:

Bradycardia, hypotension, hypoglycemia

6) WITHDRAWAL HYPERSENSITIVITY:

Acute withdrawal can lead to excess sympathetic nervous system activity in 24-48 hours due to up regulation of beta receptors.

CONTRA INDICATIONS:

1) Heart block – second and third degree

2) Cardiogenic shock-rarely associated with cardiovascular collapse in patients with compensatory tachycardia secondary to hypovolemia, cardiac failure or acute myocardial infarction.

ADVERSE EFFECTS:

Bradycardia, hypotension, deterioration (worsening) of heart failure, rebound ischemia, bronchoconstriction and fatigue.

DRUG INTERACTIONS:

1) There is 10-20% increase in digoxin levels.

2) Bradycardia and myocardial depression can occur with anaesthetic agents.

3) Morphine and Warfarin increase the steady state levels of Esmolol.

4) The duration of block by succinyl choline may be prolonged.

5) Esmolol should not be concomitantly administered with calcium channel blockers in patients with conduction abnormalities and left ventricular dysfunction.

CLINICAL USES:

1) Perioperative beta blockade

2) To attenuate intubation response.

3) Class –II anti arrhythmic effect. It is used in treatment of supraventricular

tachycardia , to convert atrial fibrillation to sinus rhythm.

4) To treat hypertension and tachycardia during surgery.

5) Myocardial ischemia

6) Thyrotoxicosis

7) Miscellaneous-glaucoma, prophylaxis of migraine, essential tremor

PHARMACOLOGY OF MAGNESIUM SULPHATE

INTRODUCTION:

Magnesium is the fourth important cation in the body. It is the second most important intracellular cation after potassium.

ROLE OF MAGNESIUM:

i. Cardiac cell membrane ion transport

ii. Enzyme activity (ATP)

iii. Physiological Antagonist of calcium

iv. Analgesia (Antagonism of NMDA receptors)

v. Systemic vasodilation

vi. Coronary vasodilation

vii. Inhibits platelet function

viii. Decreases reperfusion injury

MECHANISM OF ACTION:

It is a physiological and pharmacological blocker of N-methyl- D-aspartate receptors in neuronal tissue. It also inhibits calcium mediated neuroendocrine secretion and release of catecholamines.

It potentiates neuromuscular blockade by competing for calcium channels in pre-synaptic nerve terminal and inhibits acetyl choline release at the motor end plate.

Properties:

i. Antagonism of NMDA receptors is used for acute and chronic pain control.

ii. Used in hypotensive anaesthesia in middle ear surgeries.

iii. Inhibits catecholamine release in pneumo peritoneum in laparoscopic cholecystectomy.

iv. Decreased incidence of nausea and vomiting.

v. Decreased requirement for rescue antiemetics.

vi. Decreased incidence of post operative shivering.

vii. Decreased anaesthetic requirement.

viii. Myocardial protection.

ix. Improved control of asthma.

x. Used in tocolysis.

xi. Management of intra operative hypertensive events.

HYPOMAGNESEMIA:

Serum concentration is less than 1.6meq/litre.

Causes:

i. Chronic alcoholism

ii. Hyperalimentation

iii. Malabsorption

iv. Diuretic therapy

v. Drugs-cyclosporine

vi. Hypokalemia

Symptoms of hypomagnesemia:

- Neuromuscular manifestations resembling hypocalcemia like stridor, chvosteks sign, carpopedal spasm, trousseau's sign
- Skeletal muscle weakness
- Ventricular dysrhythmias

Treatment:

Magnesium Sulphate 1-2 g IV over 5-60 minutes

HYPERMAGNESEMIA:

Serum concentration of Magnesium >2.6mEq/litre.

Cause:

1) Parenteral Magnesium to treat PIH

2) Chronic renal failure

Symptoms:

1. Sedation
2. Myocardial depression
3. Suppression of neuromuscular transmission
4. Direct relaxant effect on skeletal and smooth muscle
5. Potentiation of non depolarizing muscle relaxants
6. Diminished deep tendon reflexes when serum Magnesium >10mEq/litre
7. Skeletal muscle paralysis, apnea and heart block when serum Magnesium >12mEq/litre

Treatment:

1) IV Fluids

2) Drug induced diuresis

3) 10%Calcium gluconate 10-15mg/kg IV

CLINICAL USES OF MAGNESIUM:

1) Pregnancy induced hypertension

Initial dose is 40-60mg/kgIV followed by an infusion 15-30mg/kg/hr to maintain therapeutic serum concentration of 4-6mEq/litre. In eclampsia it produces cerebral vasodilation, protects the blood brain barrier and has anticonvulsant properties.

Normal serum concentration---1.8-2.5mEq/l

Therapeutic level---4-7mEq/l

Loss of patellar reflex---7-10mEq/l

Respiratory depression---10-13mEq/l

Heart block---15-25mEq/l

Cardiac arrest--- >25mEq/l

2) Treatment of cardiac dysrhythmias and hypertension:

Initial dose of 2g IV administered over 5 minutes followed by infusion

1-2g/hour.

3) Cardioprotective effects

Used in patients who develop torsades de pointes type of ventricular tachycardia following acute myocardial infarction .In the cardiovascular system it produces arteriolar vasodilation, minimal venodilation, maintains cardiac filling and cardiac output.

4) Trial use in catecholamine excess states like pheochromocytoma and tetanus where magnesium limits catecholamine release are being tried.

CONTRAINDICATIONS:

1) Renal failure-since magnesium is excreted by the kidneys

2) Digitalis use

3) In patients on CNS depressants

REVIEW OF LITERATURE

KING et al., in 1951, showed that there was a marked rise in blood pressure and heart rate during laryngoscopy which was due to the mechanical stimulation of sensitive receptors in the area of epiglottis.

SANTHOSH KUMAR ,SAPNA BATHLA et al [4].,in 2003, compared the efficacy of intravenous Esmolol, Magnesium Sulphate and Diltiazem in attenuating hemodynamic stress response to laryngoscopy and intubation.

Method:

One hundred and ninety normotensive patients of ASA PS I and II, 15-55 years were randomized into four groups placebo, Esmolol, Magnesium Sulphate and Diltiazem.They were premedicated with Injection Glycopyrrolate and Diazepam forty five minutes before surgery. Anaesthesia was induced with Thiopentone and vecuronium. Esmolol 2mg/kg or Diltiazem 0.2mg/kg and Magnesium Sulphate 60mg/kg were given one minute after vecuronium and intubation was done two minutes

after the drugs. Blood pressure and heart rate were measured baseline, after drug, immediately after intubation, one minute, three minutes and five minutes after intubation

Conclusion:

Magnesium Sulphate produced tachycardia and fails to attenuate rise in heart rate. Esmolol prevented rise in heart rate, though rise in blood pressure was suppressed and not prevented by this dose of Esmolol.

MICHAEL F M JAMESet.al [5].,in 1989, showed that IV Magnesium Sulphate inhibits catecholamine release associated with tracheal intubation.

Method:

Thirty patients of ASA PS I and II, 15-51 years old were divided into two groups placebo and Magnesium Sulphate .They were premedicated with Diazepam 10mg. Anaesthesia was induced with Thiopentone and MgSo4 60mg/kg or normal saline was given. Succinylcholine was given and patient was intubated. Blood pressure, heart rate , serum magnesium and catecholamine levels were measured upto five minutes after intubation.

Result:

Magnesium Sulphate 60mg/kg pretreatment increased the heart rate by 13±3.9 beats/min. After intubation heart rate was unchanged in MgSO4 group but increased in control group. Norepinephrine and epinephrine levels increased significantly in control group compared to MgSO4 group. Blood pressure was also significantly elevated in the control group.

Conclusion:

Magnesium Sulphate significantly attenuated catecholamine mediated stress response.

A.A.VANDENBERG et al [1].,in 1997, compared Magnesium Sulphate, Esmolol , Lignocaine, Nitroglycerine and placebo for attenuation of hemodynamic responses to noxious stimuli in patients undergoing cataract surgery.

Method:

100 middle age to elderly patients with diabetes, hypertension or ischemic heart disease coming for cataract surgery were given Magnesium sulphate 40 mg/kg, Lignocaine 1.5mg/kg, Nitroglycerine 7.5 microgm/kg and Esmolol 4 mg /kg at induction of anaesthesia. Heart rate, blood pressure, rate pressure product and pressure rate quotient were measured until five minutes after intubation.

Result:

Esmolol prevented increase in heart rate and rate pressure product. Magnesium did not prevent response to laryngoscopy and intubation and there was increase in rate pressure product.

JUHI SHARMA et al [2] in 2006, compared efficacy of Esmolol and Magnesium Sulphate in attenuating pressor response to intubation in controlled hypertensive patients.

Method:

Sixty ASA II patients 45-65 years with controlled hypertension for elective surgery were allotted into three groups. They received MgSo4

40mg/kg, Esmolol 1.5mg/kg or Placebo. Anaesthesia was induced with Thiopentone and succinylcholine 1.5mg/kg. Heart rate and blood pressure was measured upto five minutes after intubation.

Result:

There was no significant difference in systolic and diastolic blood pressure between MgSo4 and Esmolol group. There was significant rise in heart rate in MgSo4 group.

D.LEE et al [30]in 2009, showed that magnesium Sulphate attenuates blood pressure response during laparoscopic cholecystectomy.

Method:

Thirty two patients undergoing laparoscopic cholecystectomy were divided into two groups placebo and MgSo4 50mg/kg. Arterial pressure, heart rate, serum magnesium, plasma renin activity, cortisol, vasopressin and catecholamine levels were measured.

Result:

Blood pressure, norepinephrine and epinephrine levels increased in placebo group.There was no significant difference in renin and cortisol levels.

Conclusion:

Magnesium Sulphate attenuates blood pressure response due to decreased release of vasopressin and catecholamine levels.

R. W.ALLEN et al [6]., in 1990, studied attenuation of pressor response to tracheal intubation in 69 patients with moderate to severe gestational proteinuric hypertension with ligocaine, Alfentanil and magnesium sulphate. No mean increase in systolic blood pressure occurred in MgSO4 group.

D.H. LEE et al [8] in 2009, showed that MgSO4 as an adjuvant during general anaesthesia for caesarean section avoided perioperative awareness and pressor response from inadequate analgesia.

K.PASTERNAK et al 9 in 2006, showed the effect of preoperative Magnesium supplementation in CABG patients. Magnesium attenuates adrenergic response to intubation.

B.UGUR et al [10] in 2007, showed that Esmolol 1.5 mg/kg prevents tachycardia and increase in rate pressure product caused by laryngoscopy and intubation in patients with tachycardia.

J.H.RYU et al [11] in2009, showed that MgSO4 provided adequate hypotensive anaesthesia and favourable post operative course with better analgesia , less shivering and less postoperative nausea and vomiting in middle ear surgeries.

E.FIGUEREDO et al [12] in 2001, did a meta analysis on the assessment of efficacy of various doses and regimes of Esmolol on haemodynamic changes induced by laryngoscopy and tracheal intubation. Various trials were performed and they measured heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure preinduction, immediately prior to intubation and post intubation. They showed that

Esmolol is effective for attenuation of adrenergic response and preferred to use it as a continuous infusion. The most effective dose was loading dose of Esmolol 500microgm/kg/min followed by an infusion of 200-300microgm/kg/minute.

K.MONTAZERI et al [13] in 2005, showed that pre treatment with different doses of MgSO4 decreased cardiovascular responses to laryngoscopy and intubation than lignocaine.

A.GUPTA et al [3] in 2009, compared Esmolol and MgSo4 for attenuation of stress response to laryngoscopy and intubation.

Method:

Sixty ASA I and II patients 20-40 years, were divided into three groups placebo, Esmolol 1.5mg/kg and Lignocaine 1.5mg/kg. Anaesthesia was induced with Thiopentone and Succinylcholine. Blood pressure and heart rate was measured upto five minutes after intubation.

Result:

Esmolol showed greater attenuation of heart rate and blood pressure than lignocaine.

MICHAEL.F.JAMES et al [32]in 2005, compared Remifentanil and MgSo4 for attenuation of hemodynamic response to electro convulsive therapy.

Method:

Twenty patients underwent anaesthesia 115 times and were allotted into three groups placebo, Magnesium Sulphate and Remifentanil. Anaesthesia was induced with Thiopentone 4mg/kg and Succinylcholine 0.5mg/kg. Remifentanil 1microgm/kg amd MsSo4 30mg/kg were given. Heart rate and blood pressure was measured 0,1, 3, 5,10 minutes after seizures.

Conclusion:

They concluded that Remifentanil and Magnesium Sulphate attenuated systolic blood pressure response to ECT. Remifentanil but not Magnesium Sulphate suppressed heart rate response to electro convulsive therapy.

RENITA MARINA PINTO et al [31]in 2007,compared hemodynamic stress response to laryngoscopy and intubation in hypertensive patients. Esmolol 2mg/kg and Magnesium Sulphate 50mg/kg was used. They concluded that both attenuated pressor response in well controlled hypertensive patients and that both are safe and effective.

DILIP KOTHARI et al [15]in 2008, compared efficacy of intravenous MgSo4 and fentanyl on circulatory changes during anaesthesia. They concluded that there was significant increase in heart rate after injection .They said that magnesium is a cheap alternative to fentanyl.

A.AHMED et al [17] in 2009,showed that MgSo4 50mg/kg attenuates stress response to laryngoscopy and intubation.

SHARMA et al [23] in 1996, compared various doses of Esmolol like 100 and 200 mg in attenuating stress response to laryngoscopy and intubation in hypertensive patients. They concluded that Esmolol 200mg

produced diastolic blood pressure less than baseline values and Esmolol 100mg is safe.

FUGHS et al [29] in 1995, studied the interaction of Magnesium Sulphate with vecuronium induced neuromuscular block. They allocated patients into two groups. One group received Magnesium Sulphate 40mg/kg in 100 ml saline as an infusion over ten minutes before induction and the other group received 100ml of saline as a placebo.

Observation:

Plasma magnesium concentrations increased from a baseline concentration

of 0.9(0.06) to 1.08 (0.07) mmol/ litre 15 min after the MgSO4 infusion ($P < 0.05$); in controls plasma magnesium concentrations were 0.86 (0.08) mmol/ litre before and 0.87 (0.6) mmol/l after saline infusion. A hot flush of the lower abdomen was reported by 21 of the 55 patients.

Conclusion:

They concluded that MgSO4, administered before vecuronium, accelerated the onset of neuromuscular block necessary for intubation of the trachea, that MgSO4 in the presence of vecuronium intensified and

prolonged neuromuscular block, and that monitoring of neuromuscular function and reduction in dose of vecuronium are required when using these two drugs in combination.

MATERIALS AND METHODS

Ninety patients of ASA physical status I and II undergoing elective ENT surgeries under general anaesthesia with endotracheal intubation were included in the study.

Patients in the age group 15-60 years of both sexes were included. It is a prospective randomized single blinded study. The study was approved by our institute ethical committee and after obtaining informed written consent from the patient, this study was conducted.

This study was done during the period from September 2012 to November 2012, in the Institute of Anaesthesiology and critical care,Rajiv Gandhi Government General Hospital, Chennai -600003.

INCLUSION CRITERIA:

- Age :15 – 60 years
- ASA : I & II
- Surgery : Elective ENT surgery
- Who have given valid informed consent.

EXCLUSION CRITERIA:

- Not satisfying inclusion criteria.
- Patients with anticipated difficult intubation
- Uncontrolled hypertension and diabetes
- History of cardiac illness
- Neuromuscular disorders
- Hepatic disease
- Renal disease
- known sensitivity to the drugs
- cerebrovascular disease
- Emergency surgery
- Bronchial asthma
- Patients on alpha and beta blockers

Materials:

- Laryngoscope blades of various sizes, bougie, oropharyngeal airway
- Drugs – Propofol, Sevoflurane, Fentanyl, Glycopyrrolate, Midazolam,Normal saline,Vecuronium, Ephedrine, Atropine, Esmolol Magnesium Sulphate, Neostigmine, Calcium gluconate and other emergency drugs.

- Monitors – ECG, NIBP, SPO2, EtCO2
- 2 cc, 5 cc and 10 cc syringes
- 18G intravenous cannula.
- Appropriate size endotracheal tubes

METHODS:

Pre anaesthetic preparation:

All the patients were admitted in the wards as inpatients and routine investigations were done.

i. Complete blood count

ii. Blood urea and sugar

iii. Serum creatinine and electrolytes

iv. chest x-ray

v. Electrocardiogram

vi. Other investigations like liver function test and coagulation profile were asked for if needed.

Anaesthetic Protocol:

Pre operative visit was done to allay the anxiety of the patients and good rapport was established with the patients.

All patients were given pre operative night sedation with tablet Alprazolam 0.25mg.

Tablet Ranitidine 150mg and tablet Metoclopramide 10mg was given with sips of water at 6 AM.

Monitors:

Induction of anaesthesia was standardized for all patients. Monitors used were NIBP, ECG, EtCo2 and pulse oximetry.

METHOD:

90 patients of age group 15 to 60 yrs of ASA physical status I and II who underwent elective ENT surgery under general anesthesia were selected and randomly allocated into 3 groups. Baseline heart rate and blood pressure was measured. They were premedicated with Inj.Glycopyrrolate 0.2mg and Inj.Midazolam 0.04mg/kg intramuscular 45 minutes before surgery. Patients were then shifted into the theatre. Inj.

Fentanyl 2µg/kg was given 5 minutes before intubation to all patients. They were induced with Inj. Propofol 2mg/kg and Vecuronium 0.1mg/kg. Nitrous oxide and Oxygen in a ratio of 66:33 and Sevoflurane 2% was given to the patient.

Group E received Esmolol 1.5mg/kg in 15ml normal saline over 15-20 seconds one minute after vecuronium and intubation was done after 2 minutes.

Group M received Inj.Magnesium Sulphate 50mg/kg in 100ml of normal saline infusion over 10 minutes before induction.

Group P received 15ml of normal saline 5 minutes before induction.

The heart rate, systolic and diastolic blood pressure and mean arterial pressure were recorded baseline, after premedication, one min after test drug, after induction, immediately after intubation, thereafter 1, 3 and 5 minutes following intubation. Laryngoscopy duration and Cormack Lehane score were noted. Any incidence of hypotension, bradycardia or arrhythmias was noted.

No surgical stimulation or change in position was permitted for five minutes after intubation. Anaesthesia was maintained with N2O:O2 in a ratio of 66:33 , vecuronium 0.01mg/kg and sevoflurane 1-2% as needed.

All patients were reversed with Inj. Glycopyrrolate 0.01mg/kg and Neostigmine 50microgm/kg and extubated after reversal of neuromuscular blockade.

PRIMARY OUTCOME MEASURES:

- Heart rate, systolic blood pressure , diastolic blood pressure, mean arterial pressure

DATA ANALYSIS:

ANOVA and Pearson chi square test were used. A p value of <0.05 was considered as statistically significant. Tukey's HSD was used to compare between the groups.

OBSERVATIONS AND RESULTS

Ninety patients under this study were allotted into three groups,

Group P- Placebo

Group E- Esmolol

Group M Magnesium sulphate

With thirty patients in each group. They comprised of both sexes in the age group 15-60 years. The demographic profile is as follows,

Characteristics		**Group P**	**Group E**	**Group M**
Age (yrs)		29.7±10.06	26.53±9.32	31.13±9.20
Sex	**Male**	11	13	11
	Female	19	17	19
Weight (kg)		56.27±8.09	52.93±10.65	52.67±8.92

Age group (Years)

Group	N	Mean	SD	ANOVA
P	30	29.70	10.06	F = 1.829
E	30	26.53	9.32	p = 0.167
M	30	31.13	9.20	

In group P, the mean age was 29.7±10.06, ranging from 16-52 yrs. In group E, the mean age was 26.53±9.32, ranging from 15-60 yrs. In group M, the mean age was 31.13±9.20, ranging from 16-45 yrs.

The p value is 0.167. Hence there is no statistical significance in age between the three groups (p value of significance is < 0.05)

Sex Distribution

Sex	**Group P**	**Group E**	**Group M**	**Total**	
Male	11	13	11	35	p = 0.829
Female	19	17	19	55	
Total	30	30	30	90	

By Pearson Chi square test p = 0.829. Hence there is no statistical significance in sex between the three groups.

Weight Distribution (kg)

Group	N	Mean	SD	ANOVA
P	30	56.27	8.09	F = 1.401 p = 0.252
E	30	52.93	10.65	
M	30	52.67	8.92	

In group P, the mean weight was 56.27±8.09, ranging from 38-71kg. In group E, the mean weight was 52.93±10.65, ranging from 35-70kg. In group M, the mean weight was 52.67±8.92, ranging from 35-74kg.

p value = 0.252. Hence there is no statistical significance in weight distribution between the three groups.

Baseline Hemodynamic Parameters

Basal Heart rate (beats/min)

Group	N	Mean	SD	ANOVA
P	30	79.80	14.44	F = 3.690 p = 0.112
E	30	86.67	9.79	
M	30	81.47	14.47	

p value = 0.112. Hence there is no statistical significance in basal heart rate between the three groups.

Basal systolic pressure (mmHg)

Group	N	Mean	SD	ANOVA
P	30	125.73	10.72	F = 0.690 p = 0.504
E	30	123.13	8.69	
M	30	125.90	11.12	

p value = 0.504. Hence there is no statistical significance in basal systolic pressure between the three groups.

Basal diastolic pressure (mmHg)

Group	N	Mean	SD	ANOVA
P	30	81.33	6.70	F = 0.348 p = 0.707
E	30	80.00	6.50	
M	30	81.33	8.12	

p value = 0.707. Hence there is no statistical significance in basal diastolic pressure between the three groups.

Basal mean arterial pressure (mmHg)

Group	N	Mean	SD	ANOVA
P	30	95.93	7.46	F = 0.613 p = 0.544
E	30	94.07	6.60	
M	30	96.00	8.81	

p value = 0.544. Hence there is no statistical significance in basal mean arterial pressure between the three groups.

HEART RATE CHANGES (beats / min)

Group	P	E	M	p Value
Baseline	79.80±14.44	86.67±9.79	81.47±14.47	0.112
After Premedication	79.83±13.05	86.63±14.30	84.53±16.50	0.191
After Test Drug	77.30±11.30	77.70±13.14	93.27±18.45	0.001
After Induction	72.27±13.26	82.20±15.00	78.33±11.31	0.017
Immediately After Intubation	103.63±12.31	90.67±13.93	97.40±12.57	0.001
1 minute After Intubation	95.87±12.86	87.27±13.48	92.97±10.38	0.026
3 minutes After Intubation	87.83±12.50	84.30±12.79	90.07±9.82	0.167
5 minutes After Intubation	81.73±13.66	81.60±13.31	86.00±9.61	0.296

SYSTOLIC BLOOD PRESSURE CHANGES (mmHg)

Group	**P**	**E**	**M**	**p Value**
Baseline	125.73±10.72	123.13±8.69	125.90±11.12	0.504
After Premed	123.47±10.54	120.67±9.05	120.30±9.56	0.391
After Test Drug	118.70±13.64	100.10±9.60	112.27±11.65	0.001
After Induction	109.97±13.63	107.70±9.23	98.97±9.74	0.001
Immediately After Intubation	138.57±17.25	114.43±8.76	124.90±17.76	0.001
1 minute After Intubation	128.73±17.04	107.60±9.23	116.80±15.66	0.001
3 minutes After Intubation	119.13±16.16	102.60±9.61	109.80±14.75	0.001
5 minutes After Intubation	111.23±14.34	100.20±9.44	105.47±10.84	0.002

DIASTOLIC BLOOD PRESSURE CHANGES (mmHg)

Group	**P**	**E**	**M**	**p Value**
Baseline	81.33±6.70	80.00±6.50	81.33±8.12	0.707
After Premed	78.40±6.89	76.33±5.82	77.30±8.61	0.541
After Test Drug	76.93±7.38	61.90±8.22	69.70±9.63	0.001
After Induction	71.27±10.28	67.63±8.05	59.30±9.79	0.001
Immediately After Intubation	93.60±12.49	74.73±8.29	83.13±16.74	0.001
1 minute After Intubation	85.77±12.16	67.53±9.97	76.17±15.94	0.001
3 minutes After Intubation	79.80±12.27	62.63±10.19	71.07±14.29	0.001
5 minutes After Intubation	72.63±10.20	60.00±9.93	67.10±12.20	0.001

MEAN ARTERIAL PRESSURE CHANGES (mmHg)

Group	**P**	**E**	**M**	**p Value**
Baseline	95.93±7.46	94.07±6.60	96.00±8.81	0.544
After Premed	93.20±7.49	90.63±6.76	91.30±8.73	0.412
After Test Drug	90.77±8.56	74.30±7.78	83.30±9.84	0.001
After Induction	84.00±10.36	81.00±7.34	72.23±9.34	0.001
Immediately After Intubation	108.10±14.19	87.70±8.01	96.87±16.72	0.001
1 minute After Intubation	100.10±13.49	80.67±9.19	89.97±15.64	0.001
3 minutes After Intubation	92.77±13.17	75.70±9.01	84.00±13.79	0.001
5 minutes After Intubation	85.30±11.17	73.30±8.69	79.50±10.73	0.001

MMS GRADE(MODIFIED MALLAMPATTI SCORE)

Grade	Group P	Group E	Group M	Total	Chi Square Test
l	22	24	24	70	p = 0.695
ll	7	6	6	19	
lll	1	0	0	1	
Total	30	30	30	90	

CORMACK LEHANE GRADE

Grade	Group P	Group E	Group M	Total	Chi Square Test
l	27	22	19	68	p = 0.166
ll a	3	6	8	17	
ll b	0	2	3	5	
Total	30	30	30	90	

LARYNGOSCOPY DURATION

Group	N	Mean	SD	ANOVA
P	30	13.23	2.34	F = 1.070
E	30	12.87	2.45	p = 0.348
M	30	13.83	2.93	

SIDE EFFECTS

HYPOTENSION

Grade	Group P	Group E	Group M	Total	Chi Square Test
Yes	0	1	4	5	
No	30	29	26	85	p = 0.133
Total	30	30	30	90	

BRADYCARDIA

Grade	Group P	Group E	Group M	Total	Chi Square Test
Yes	0	1	1	2	
No	30	29	29	88	p = 0.600
Total	30	30	30	90	

ARRHYTHMIA

Grade	Group P	Group E	Group M	Total
Yes	0	0	0	0
No	30	30	30	90
Total	30	30	30	90

There was no incidence of laryngospasm, bronchospasm or prolongation of neuromuscular blockade.

DISCUSSION

Laryngoscopy and intubation can produce haemodynamic stress response charecterised by hypertension and tachycardia. It can also produce increase in intracranial pressure. No drug can attenuate this response completely. Many drugs have been reported to attenuate this response.

A.A.Vandenberg et al [1] in October 2003, studied attenuation of haemodynamic response in Magnesium Sulphate pretreated patients undergoing cataract surgery. They compared Esmolol and MgSO4.They concluded that Esmolol 4mg/kg prevented increase in heart rate and rate pressure product. Mg SO4 40 mg/kg did not prevent response to laryngoscopy and intubation.

SapnaBathla,Santhoshkumar et al [4] in 2003, compared efficacy of IV Esmolol Diltiazem and Magnesium Sulphate, in attenuating haemodynamic stress response to laryngoscopy and intubation. Their study showed that MgSo4 produced tachycardia and failed to attenuate rise in heart rate. Esmolol prevented rise in heart rate though rise in blood pressure was suppressed but not prevented.

In our study, comparison of Esmolol 1.5mg/kg, MgSo4 50mg/kg and placebo was done in attenuating haemodynamic stress response to laryngoscopy and intubation. The data was analysed using Microsoft Excel. Haemodynamic variables were represented by mean and standard deviation. Statistical significance was assessed by use of ANOVA and Pearson chi square test. TUKEYS HSD was applied to evaluate inter group comparisons .

P vaue less than 0.05 was considered as statistically significant.

HEART RATE CHANGES:

The baseline heart rate of all groups did not have any statistical significance since p value was 0.112.

After the test drug there was significant decrease in heart rate in Esmolol group compared to the placebo, whereas there was increase in heart rate in Magnesium sulphate group. This result was also observed by **M F M James et al** [5] in 1989 which showed that Magnesium sulphate pretreatment increased heart rate by 13±3.9 beats per minute. In our study heart rate increased by 11.8±3.98 beats per minute.

After induction heart rate in all three groups reduced significantly compared to baseline.

Immediately after intubation, heart rate increased in all three groups. p=0.001(statistically significant).But Esmolol group produced just a little increase in heart rate (4±4.4 beats/min). Esmolol produced least increase in heart rate compared to baseline.

Both MgSO4 and placebo group produced increase in heart rate response but by Tukey HSD it was not significant since both groups had increased heart rate by approximately 15-20 beats from baseline.

One minute after intubation heart rate increased significantly between the groups. p=0.026.Esmolol produced least increase in heart rate. Both group P and M produced increase in heart rate but by Tukey HSD it was not significant.

Three minutes after intubation, heart rate between the groups was not statistically significant. p=0.167. In Group E heart rate decreased to less than baseline. Heart rate changes between group P and E was not significant, but between P and M and group E and M it was significant. Heart rate continued to be high in group M.

At five minutes after intubation, there was no statistical difference p=0.296. The mean heart rate in Group E was less than baseline but marginally elevated in group P and M.

Juhi Sharma et al [2] when comparing Esmolol and Magnesium Sulphate in attenuating pressor response in controlled hypertensive patients stated that Magnesium Sulphate produces increase in heart rate.

G.D. Puri et al [16] showed increase in heart rate after MgSO4 injection and further lncrease with intubation in MgSO4 pretreated coronary artery disease patients.

B.Ugur et al [10] in 2007, showed that Esmolol 1.5mg/kg prevented tachycardia and increase in rate pressure product caused by laryngoscopy and intubation in patients with tachycardia.

This result was in accordance with **A.Gupta et al** [3] in 2009 who showed greater attenuation of heart rate and blood pressure response with Esmolol 1.5mg/kg than Lignocaine.

In this study, Esmolol produced least increase in heart rate. Even three minutes after intubation the heart rate continued to be high in group M.

The heart rates started coming towards baseline value five minutes after intubation.

SYSTOLIC BLOOD PRESSURE CHANGES

The baseline systolic blood pressure between the three groups was not statistically significant p=0.504.

After test drug there was significant fall in blood pressure between the three groups p=0.001.Group E produced the maximum fall in SBP followed by group M and P. There was no significance between group P and M. But there was significance between group P and E and group E and M.

After induction there was significant fall in BP between the three groups p=0.001.

Immediately after intubation there was significant in SBP changes between the groups. p=0.001.Group E produced fall in SBP compared to baseline. In group M the systolic blood pressure was comparable to the baseline and in group P it was higher than the baseline.

One minute after intubation, systolic blood pressure was decreased in group E and M. Group E produced more fall in BP than group M. There was statistical significance between the three groups p=0.001.

Three minutes after intubation there was significant change in SBP between the groups. p=0.001.There was no significance between Group E and M but there was significance between group P and E and group P and M. In group E there was significant fall in BP compared to the other groups.

Five minutes after intubation, there was significance in SBP changes between the groups p=0.001. There was statistical significance between group P and E but no significance between group P and M and group E and M.

These results are in accordance with **A.Rathore et al** [27] who stated that Esmolol 150mg caused significant blunting of rise in systolic blood pressure. But in this study we used Esmolol 1.5mg/kg which was enough to blunt intubation response.

Sharma et al [23] showed that Esmolol 100mg was safe to blunt hemodynamic stress response.

R.W.Allen et al [6] in 1990, showed no mean increase in systolic blood pressure to tracheal intubation in hypertensive proteinuric pregnant patients given Magnesium Sulphate.

Overall Group M showed less hypertensive response compared to group P, but group E had the least hypertensive response.

DIASTOLIC BLOOD PRESSURE CHANGES

The baseline diastolic blood pressure between the three groups was not statistically significant p=0.707.

After test drug, there was significant fall in BP between the three groups.p=0.001

After induction, Magnesium Sulphate produced more fall in diastolic blood pressure than Esmolol. There was statistical significance between group P and M and between group E and M.

Immediately after intubation, one minute, three minutes and five minutes after intubation there were significant changes in diastolic blood pressure between the groups p=0.001.Diastolic blood pressure was increased more in group P and to a less extent in group M, but Group E showed fall in BP compared to the baseline.

K.Montazeri et al [13],showed that pretreatment with different doses of Magnesium Sulphate suppressed blood pressure response to laryngoscopy and intubation.

A.Ahmed et al [17].,showed that MgSo4 50mg/kg attenuated stress response to laryngoscopy and intubation.

Juhi Sharma et al [2] stated that there was no significant difference in systolic and diastolic blood pressure between MgSo4 and Esmolol group.

Thus this study showed that Esmolol and Magnesium Sulphate produced significant fall in diastolic blood pressure after induction and intubation compared to placebo group.

MEAN ARTERIAL PRESSURE CHANGES

The baseline mean arterial blood pressure between the three groups was not statistically significant p=0.544

After test drug, there was statistical significance of fall in mean arterial pressure between the three groups. p=0.001

After induction, group M produced more fall in BP than group E and P.

Immediately after intubation, one minute and three minutes after intubation there were significant changes in mean arterial blood pressure between the groups p=0.001.Fall in mean arterial blood pressure was more in group E followed by group M. In group P there was an increase in MAP.

K.Pasternak et al [9].,showed that Magnesium Sulphate attenuated adrenergic response to tracheal intubation in CABG patients.

Sharma et al [23]showed that Esmolol 200mg blunts hemodynamic stress response to tracheal intubation in treated hypertensive patients and the hemodynamic variables were lower than the basal values. Since our study was conducted on non hypertensive patients Esmolol 1.5mg/kg was sufficient to blunt stress response and produced hemodynamic variables lower than baseline values.

Tetsuro Kagawa et al [28].,showed that IV MgSo4 significantly suppressed mean arterial pressure and rate pressure product at the time of tracheal intubation. They also stated that no sedative effect was observed. Even in our study we observed no sedative effect in group M patients.

Five minutes after intubation, there was statistical significance in mean arterial pressure changes between the groups .p=0.001. By Tukey's HSD there was significance between group P and E but no significance between group E and M or group P and M. Group E had MAP less than the baseline.

Esmolol and Magnesium Sulphate produced significant fall in mean arterial pressure compared to placebo group.

SIDE EFFECTS

One patient in group E and four patients in group M had hypotension (MAP<60mmHg) .One patient in group M and one patient in group E had bradycardia (HR<60/min).There were no incidences of bronchospasm or laryngospasm and arrhythmia in any group. There were no cases of prolonged neuromuscular blockade or delayed recovery in any group. One patient in group M had complaints of hot flush in the lower abdomen when Magnesium Sulphate was being infused which was also observed in the study by **Fughs et al.**

This study shows that Esmolol attenuates increase in heart rate and blood pressure following laryngoscopy and intubation. Magnesium Sulphate is also useful in attenuating blood pressure changes but produces tachycardia. Hence Esmolol is useful in attenuating stress response followed by Magnesium Sulphate compared to a placebo.

SUMMARY

This randomized prospective single blinded study was designed to evaluate efficacy of Esmolol and Magnesium sulphate in attenuating hemodynamic stress response to laryngoscopy and intubation .Ninety patients of ASA PS I and II were randomly allocated into three groups of thirty each.

P---received normal saline

E—Esmolol 1.5mg/kg

M—Magnesium Sulphate 50mg/kg

The following observations were made

1) Group E showed maximum attenuation of heart rate and blood pressure.

2) Group M also showed significant attenuation of blood pressure response but produced tachycardia on infusion of the drug. Heart rate response was not statistically significant compared to group E.

3) All patients recovered well.

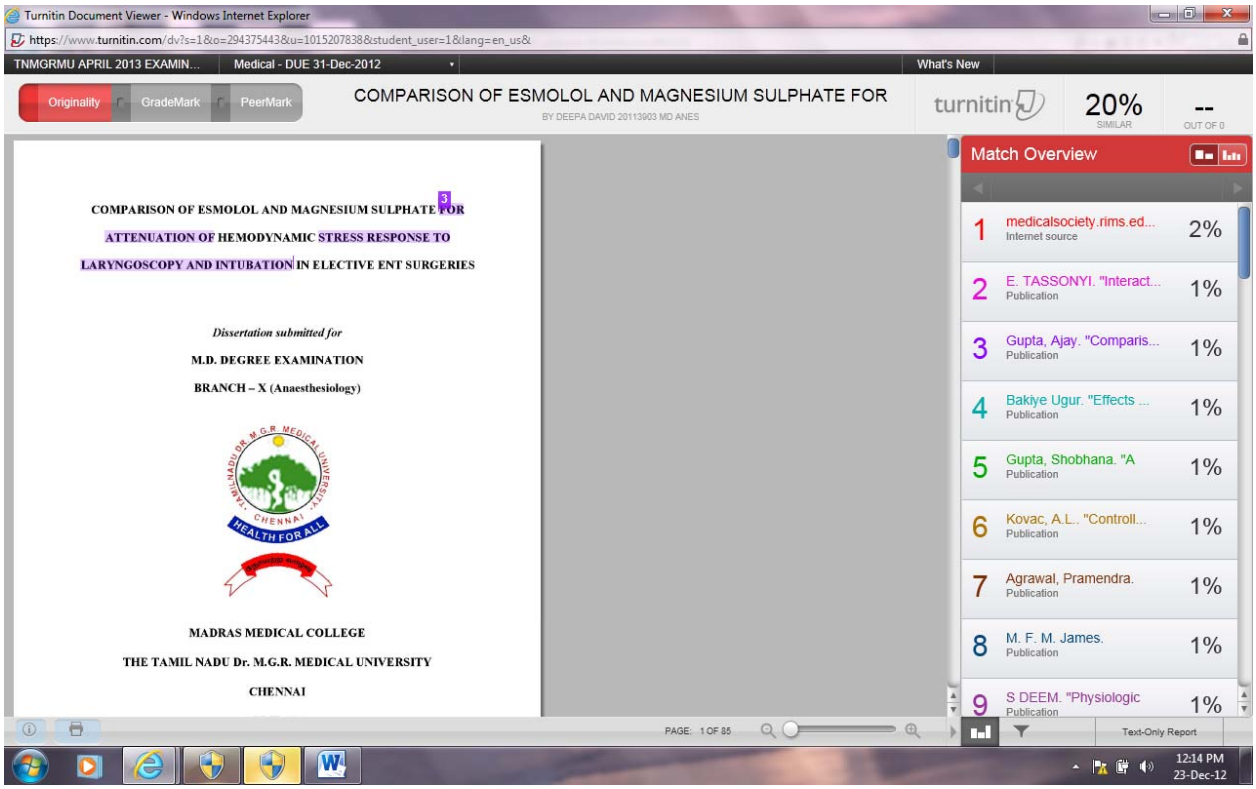
4) Incidence of side effects was not significant between the groups.

CONCLUSION

From this study, it is concluded that hemodynamic changes to laryngoscopy and intubation can be attenuated by giving intravenous Esmolol 1.5mg/kg.

Esmolol is effective in blunting the response followed by Magnesium Sulphate which blunts the hypertensive response but produces tachycardia during infusion of the drug.

Placebo was ineffective in blunting hemodynamic stress response to laryngoscopy and intubation.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MAGNESIUM SULPHATE GROUP

S. No	NAME	AGE	SEX	WEIGHT	IP NO	BASELINE				AFTER PREMED				AFTER TEST DRUG				AFTER INDUCTION				IMMED AFTER INTUBA				1MIN AFTER INTUBA				3 MIN AFTER INTUBA				5 MIN AFTER INTUBA				TMMS	CL GRA	LARYNGOSCOP	SIDE EFFECTS		
						HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP			DURN(SECS)	HYPOTN	BRADYARRHYTHMIAS
1	VIMALANAND	30	M	58	61516	76	120	76	91	78	120	75	90	82	110	72	85	70	105	70	82	81	108	72	82	79	105	71	82	76	103	70	81	70	101	63	76	I	I	9	N	N	N
2	DHANALAXMI	36	F	55	66322	86	136	84	101	92	126	84	98	96	119	77	91	74	108	63	78	106	153	109	124	97	130	95	107	91	116	81	93	87	114	78	90	I	I	10	N	N	N
3	DILLIKUMAR	34	M	50	63449	62	108	71	80	68	109	68	78	73	113	68	77	67	100	56	66	83	114	75	85	91	121	87	98	85	117	77	86	80	106	87	76	I	I	13	N	N	N
4	PRAKASAM	19	M	55	63455	80	136	86	103	97	124	85	98	106	120	77	88	76	93	46	62	105	130	79	96	100	134	80	98	93	106	63	77	88	101	53	69	II	I	12	N	N	N
5	UMA	35	F	47	64679	105	136	86	103	126	122	75	85	125	125	73	84	102	108	57	68	113	123	82	93	103	114	68	78	102	110	67	75	100	111	64	74	I	I	15	N	N	N
6	DIVYABARATHI	16	F	48	65996	104	130	82	98	118	117	71	86	137	113	70	84	95	98	60	73	113	119	80	93	107	90	58	69	104	80	48	59	96	87	53	64	I	IIA	13	Y	N	N
7	BHUVANESWARI	37	F	57	65832	118	132	88	103	121	126	81	96	113	117	76	90	83	108	71	83	113	130	94	106	94	115	74	88	92	115	74	88	91	100	59	73	I	I	10	N	N	N
8	ANJALIDEVI	36	F	67	67736	90	142	90	107	91	115	82	93	90	123	81	95	81	105	66	79	104	150	107	121	102	154	108	123	98	150	104	119	95	124	77	93	I	IIB	18	N	N	N
9	VELU	33	M	74	68003	84	128	86	100	74	121	85	97	86	117	80	92	75	104	71	82	81	124	86	99	82	123	83	96	82	119	82	94	80	116	78	91	I	IIA	19	N	N	N
10	DHANASEKAR	18	M	57	65013	54	118	68	85	56	114	64	81	62	104	62	76	73	93	54	67	76	99	66	77	75	99	65	76	78	98	64	75	80	96	61	73	I	I	12	N	N	N
11	MURALI	44	M	45	68184	76	118	80	93	74	114	78	90	82	101	71	81	82	101	72	82	108	150	117	128	105	140	102	115	103	107	83	91	90	103	80	88	I	IIA	15	N	N	N
12	SANDHYA	17	F	55	68115	96	130	88	102	90	144	100	115	98	141	93	109	88	130	85	100	108	139	99	112	92	129	90	103	88	128	85	99	80	124	84	97	II	IIB	18	N	N	N
13	KARTHICK	45	M	50	69857	67	110	78	89	65	106	66	79	83	100	54	69	72	96	44	61	96	113	58	76	87	109	54	72	86	104	49	67	85	101	46	64	II	IIA	22	N	N	N
14	KAUSALYA	19	F	55	69083	78	110	68	82	90	109	65	80	106	106	57	73	93	89	42	58	106	107	63	78	105	103	53	70	102	99	50	66	98	101	47	65	II	IIA	12	Y	N	N
15	MARIMUTHU	35	F	42	70600	60	134	92	106	64	130	90	103	77	107	72	84	66	95	62	73	96	143	116	125	94	140	112	121	88	135	104	114	80	117	93	101	I	I	12	N	N	N
16	RAVI	42	M	50	73569	64	126	90	102	62	120	86	97	58	116	74	88	52	78	52	61	86	118	80	93	84	112	78	89	78	109	74	86	72	98	64	75	I	IIA	13	Y	Y	N
17	PRITAKUMARI	36	F	35	74890	78	105	71	82	80	98	68	78	90	92	61	71	82	89	57	68	108	117	86	96	106	113	83	93	104	112	83	93	102	98	70	79	I	I	12	N	N	N
18	KOMALA	38	F	55	76925	84	146	92	110	90	128	86	100	115	120	72	88	86	99	57	71	105	149	95	113	99	120	76	104	91	106	68	81	84	106	67	80	I	I	13	N	N	N
19	TAMILSELVI	29	F	50	76902	72	130	80	97	80	127	80	96	86	123	78	93	76	115	75	88	104	150	103	119	103	142	99	113	102	133	93	106	100	130	91	104	I	I	12	N	N	N
20	CHINNAKANU	40	F	70	76639	80	122	74	90	78	120	68	85	74	109	67	81	70	106	63	77	98	160	103	122	89	139	89	106	84	127	82	97	83	121	72	88	I	IIB	15	N	N	N
21	KANIYAMMAL	35	F	45	77742	64	118	80	93	68	103	68	80	64	96	65	75	59	93	63	73	73	105	73	84	69	98	69	79	68	90	64	73	63	89	63	72	I	IIA	16	N	N	N
22	SARRADHA	23	F	62	78179	84	138	80	95	82	127	76	93	82	106	58	74	71	94	52	66	70	97	53	68	72	106	66	79	74	106	67	80	75	113	70	84	I	I	10	N	N	N
23	GEETHA	23	F	43	78192	76	114	80	91	90	113	76	88	103	93	59	70	74	90	54	66	101	98	68	78	95	97	68	78	91	80	60	67	84	84	60	68	I	I	14	Y	N	N
24	VELAN	32	M	50	77753	70	140	98	112	80	131	89	103	95	125	86	99	75	97	56	70	82	109	73	85	84	107	72	84	86	105	68	80	78	101	69	80	II	I	11	N	N	N
25	BASHEERA	20	F	49	78103	84	122	86	98	80	119	73	88	90	105	53	70	81	98	47	64	94	114	72	86	100	112	53	73	98	110	53	72	88	104	50	68	I	I	14	N	N	N
26	MADHINA	24	F	40	81435	100	134	84	101	96	130	83	99	110	129	80	96	102	98	59	72	108	122	86	98	105	121	75	90	103	108	66	80	100	105	65	78	i	i	14	N	N	N
27	SUMATHY	31	F	45	79053	86	110	66	81	84	124	79	94	101	100	63	75	73	87	48	61	101	118	71	87	92	105	62	76	84	104	63	77	85	101	63	76	I	IIA	15	N	N	N
28	KAUSALYA	19	F	47	70605	82	120	72	88	78	118	72	87	96	98	60	73	79	90	54	66	98	130	76	94	90	112	63	79	84	109	62	86	82	108	61	87	I	I	14	N	N	N
29	MOORTHY	43	M	61	76471	90	128	76	93	92	124	72	89	114	118	64	82	82	98	60	73	98	130	74	93	94	109	68	82	92	105	65	78	90	104	63	77	I	I	15	N	N	N
30	SEKAR	45	M	63	72732	94	136	88	104	92	130	74	93	104	122	68	86	91	104	63	77	107	128	78	95	94	105	64	78	95	103	63	80	94	100	62	75	II	I	17	N	N	N

ESMOLOL GROUP

NAME	AGE	SEX	WEIGHT	IP NO	BASELINE			
					HR	SBP	DBP	MAP
VIMALKUMAR	21	M	62	61125	78	130	80	97
LAKSHMIKANT	23	M	53	65306	86	120	82	95
AMMU	27	F	60	62668	104	112	80	91
PARIMALA	31	F	68	62557	68	120	78	92
RAMANI	21	M	55	63994	98	130	84	99
INBASEKARAN	31	M	65	58498	84	124	70	89
MYTHILI	20	F	46	62219	82	122	74	90
RAJESWARI	25	F	48	60820	86	126	80	96
GEETHA	22	F	46	62875	104	118	76	87
MUSINAPARVIN	28	F	62	62223	76	102	70	80
PARVIN BANU	22	F	43	63495	90	120	80	93
CHINAPONNU	35	F	50	64993	74	130	90	103
BALACHANDRAN	28	M	58	65027	76	126	84	98
TAMILMOZHI	18	F	37	65691	124	100	70	80
SELVI	38	F	62	66320	90	120	76	91
KALAIVANI	22	F	38	64673	86	116	88	97
NAVINKUMAR	15	M	35	63457	107	130	84	99
UMA	18	F	37	60214	108	122	78	93
MOHANAPRIYA	16	F	37	65785	105	124	84	97
GUNASEKAR	19	M	54	69503	92	134	80	98
NABISHA	27	F	52	70607	102	128	86	100
ELAVARASAN	26	M	66	71868	86	144	100	115
JAYAKUMAR	60	M	70	73577	76	132	80	97
RAJATHI	30	F	45	73479	78	124	78	93
AJITKUMAR	16	M	44	74918	80	128	74	92
RAMESHKUMA	28	M	65	76471	78	116	72	87
NAGAMMAL	38	F	62	73917	86	126	80	95
RAJKUMAR	40	M	64	74570	88	124	82	96
KAVIARASAN	19	M	45	72153	88	126	74	91
SUSEELA	32	F	59	72063	88	120	86	91

AFTER PREMED				AFTER TEST DRUG				AFTER INDU
HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR
70	117	67	77	69	101	54	66	70
82	118	82	94	71	96	65	75	73
90	113	78	90	93	90	69	76	104
73	124	71	89	58	85	48	60	71
94	124	83	97	71	91	54	66	71
72	126	75	92	64	99	53	68	66
81	113	74	84	67	103	57	71	77
98	124	85	98	83	108	77	87	102
106	111	63	74	92	105	61	73	103
80	96	68	77	67	85	51	62	75
83	112	75	87	73	96	62	73	67
72	128	83	98	74	108	78	88	73
71	125	81	96	69	100	59	73	62
118	119	79	92	109	117	82	94	110
86	118	74	89	67	103	57	72	77
90	119	86	97	84	107	70	82	90
124	131	81	98	107	88	58	68	118
96	120	78	92	89	103	69	80	92
104	120	77	91	91	111	69	83	98
100	142	72	95	75	110	53	72	84
100	121	77	92	99	98	63	75	98
67	143	81	102	67	125	60	82	64
75	126	77	93	58	90	60	70	62
90	126	83	97	71	93	62	72	74
78	124	70	88	84	112	64	80	88
74	110	66	81	80	104	62	76	81
74	120	78	92	71	93	57	69	76
80	120	78	92	72	98	68	78	76
84	114	72	86	74	94	63	73	79
87	116	76	89	82	90	52	65	85

JCTION			IMM AFTER INTUBATION				1`MIN AFTER INTUBAT	
SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP
120	65	83	83	116	66	76	80	106
93	67	76	88	108	77	87	84	108
94	71	79	105	98	71	80	104	96
98	57	71	65	112	61	78	63	89
111	71	84	78	105	69	81	84	105
114	71	85	68	103	63	76	63	106
110	57	75	80	107	66	81	78	104
100	75	83	103	120	87	98	96	100
102	61	72	108	112	77	87	106	104
92	57	69	87	110	78	89	86	104
115	80	92	84	114	72	86	84	97
120	78	92	80	109	77	88	76	98
113	66	82	76	114	72	86	76	110
117	80	92	114	103	67	79	112	100
110	57	75	80	107	66	80	78	104
113	75	88	96	115	74	87	88	108
122	78	93	114	112	68	83	110	106
102	66	78	104	118	84	95	100	114
119	74	89	92	111	69	83	92	105
123	58	80	97	134	73	93	96	133
111	76	89	110	125	85	98	108	112
99	54	69	99	136	98	111	83	133
94	62	73	70	111	74	86	65	109
106	73	84	76	114	79	91	73	111
113	69	84	92	122	75	91	88	115
109	66	80	94	111	66	81	92	108
107	70	82	91	116	79	91	90	104
99	69	79	78	127	84	98	74	119
107	73	84	106	118	81	93	98	107
98	53	68	102	125	84	98	91	113

TION			3 MIN AFTER INTUBATION				5 MIN AFTER INTUBATION		
DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	
53	67	78	94	50	65	76	95	50	
77	87	80	105	71	82	73	102	68	
70	79	103	93	69	77	102	80	56	
41	57	60	90	42	58	57	93	43	
69	81	77	92	62	72	72	92	60	
55	72	64	98	53	64	62	99	52	
53	66	75	101	50	66	74	104	43	
74	83	91	95	70	78	74	96	72	
77	85	102	95	48	61	102	90	42	
73	83	85	97	71	80	83	87	59	
57	69	82	92	54	67	80	94	49	
60	73	75	86	56	66	74	85	56	
66	81	75	105	61	75	74	101	57	
65	77	110	94	59	71	110	89	56	
53	70	75	101	50	67	74	104	53	
64	79	87	103	51	68	86	98	53	
64	78	104	98	63	75	102	97	62	
81	92	102	112	79	90	98	108	75	
69	81	92	106	58	74	90	107	58	
71	92	94	130	56	81	92	125	52	
78	89	98	111	78	89	92	110	73	
83	100	76	118	73	88	67	107	64	
73	85	64	107	73	84	64	106	72	
75	87	72	109	72	84	71	105	71	
65	82	84	111	60	77	78	107	59	
64	79	88	108	66	80	90	107	65	
74	84	84	100	70	80	82	98	68	
81	94	72	118	80	93	69	116	78	
66	80	90	103	65	78	88	105	72	
75	88	90	106	69	81	92	99	62	

MAP	MMS	CL GRADE	LARYNOSCOPY DURN (secs)	SIDE EFFECTS HYPOTENSION	BRADY	ARRHYTH
65	I	I	16	N	N	N
79	I	I	11	N	N	N
64	I	II A	12	N	N	N
60	I	IIA	13	Y	Y	N
71	I	I	11	N	N	N
63	I	I	10	N	N	N
72	I	I	10	N	N	N
80	I	I	11	N	N	N
52	I	I	9	Y	N	N
68	I	I	13	N	N	N
64	I	I	10	N	N	N
66	I	I	13	Y	N	N
72	I	IIA	12	N	N	N
67	I	I	12	N	N	N
70	I	IIA	12	N	N	N
68	I	I	14	N	N	N
74	I	I	11	N	N	N
86	I	I	11	N	N	N
74	I	I	11	N	N	N
76	I	I	13	N	N	N
85	II	IIB	16	N	N	N
78	II	IIA	18	N	N	N
83	II	IIB	16	N	N	N
82	II	I	14	N	N	N
75	I	I	12	N	N	N
79	I	I	14	N	N	N
78	II	I	16	N	N	N
91	II	IIA	19	N	N	N
83	I	I	12	N	N	N
74	I	I	14	N	N	N

CONTROL GROUP

SL NO	NAME	AGE	SEX	WEIGHT	IP NO	BASELINE HR	SBP	DBP
1	MUTHUVEDI	43	F	50	73764	76	120	80
2	BHAVANI	17	F	38	60818	120	122	80
3	ADHIYAMAN	19	M	55	63937	66	136	85
4	KALISHA	40	M	55	65305	52	136	80
5	VENKATESAN	40	M	63	63612	70	122	86
6	SELVI	40	F	57	63174	76	120	82
7	MAGDALINEMARY	32	F	55	63416	88	126	86
8	MUDALIDHARAN	37	M	65	65830	118	140	86
9	MUTHUBHARATHI	28	M	55	67151	88	128	92
10	THIRUMAL	20	M	55	70714	64	142	84
11	BALAJI	17	M	40	70759	66	96	60
12	GEETHA	32	F	57	73083	78	134	82
13	GUNASUNDARI	19	F	51	73595	82	120	72
14	VASANTHI	25	F	64	72061	76	132	80
15	VINODKUMAR	17	M	58	73111	72	120	74
16	SARANYA	18	F	50	79048	64	110	75
17	DEVI	16	F	47	78570	86	126	84
18	SHAHINA	30	F	65	78566	84	126	84
19	SUSEELA	32	F	65	75486	86	144	86
20	SHANTHI	45	F	71	73123	78	143	87
21	VENDA	35	F	50	73081	78	130	84
22	BASHEERA	25	F	45	69849	78	120	80
23	LATHA	33	F	63	78217	78	130	90
24	NAVEENRAJ	18	M	65	80691	88	110	70
25	VIMALA	35	F	58	79023	58	111	77
26	VENNILA	40	F	45	78175	74	126	86
27	LAKSHMI	32	F	62	80144	84	126	84
28	SELVAM	52	M	64	81123	90	130	86
29	MANI	19	M	62	78342	92	128	86
30	BHUVANESWARI	35	F	58	68756	84	118	72

AFTER PREMED				AFTER TEST DRUG				AFTER INDU	
MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR
93	86	117	79	92	78	94	69	77	80
94	114	120	78	92	99	100	67	78	104
97	70	135	81	92	63	117	73	85	64
99	52	134	80	98	56	113	83	93	55
98	68	114	82	93	58	96	73	81	57
96	82	115	70	85	71	114	69	84	68
99	84	130	88	102	78	114	84	94	75
104	115	135	85	102	109	145	96	112	107
104	74	127	88	101	75	125	87	100	74
103	64	136	84	101	67	124	73	90	62
72	75	95	59	70	74	103	69	80	74
99	93	129	83	98	92	125	82	96	73
88	80	120	77	91	74	114	77	89	68
97	74	130	78	95	74	136	77	97	73
89	75	125	66	86	76	126	71	89	66
87	62	105	65	78	60	106	68	81	54
98	85	121	80	95	84	120	81	95	72
98	80	127	74	92	78	124	73	90	64
105	78	145	80	102	74	146	73	97	75
106	76	142	82	102	75	141	83	102	70
99	85	125	82	96	79	123	80	94	52
94	78	119	79	92	83	116	77	90	68
103	93	122	84	97	89	130	92	105	73
83	80	110	70	83	76	110	74	86	59
88	66	112	73	86	72	93	66	75	74
99	72	122	84	97	74	118	80	93	77
98	80	124	80	95	78	125	80	95	74
101	86	124	82	96	85	125	78	94	102
100	88	124	85	98	90	122	83	96	82
87	80	120	74	89	78	116	70	85	72

JCTION			IMMED AFTER INTUBATION				1 MIN AFTER INTUBATION		
SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	DBP
94	70	78	93	117	84	95	88	98	71
103	69	80	121	121	78	92	120	111	73
122	74	85	95	128	86	99	92	127	83
114	83	93	85	163	112	129	80	166	112
97	74	82	105	147	102	117	83	116	86
111	67	82	75	126	81	96	67	101	65
112	83	93	120	148	111	123	115	140	102
144	96	112	138	158	103	121	128	136	92
96	72	80	106	132	95	107	100	130	92
102	67	79	92	151	99	116	73	141	90
107	65	79	106	115	79	91	100	107	68
97	61	73	105	140	98	112	109	140	97
109	58	75	87	124	81	95	86	122	81
105	64	78	107	146	96	113	94	140	92
93	60	71	99	121	76	81	95	107	61
93	52	66	108	124	89	101	98	103	73
106	74	85	116	150	108	122	98	150	98
116	70	85	104	150	108	122	98	141	100
128	59	82	99	153	92	112	92	135	77
136	90	105	99	182	106	131	92	155	98
117	66	83	117	163	110	128	100	153	98
108	72	84	98	127	82	96	84	119	80
110	77	88	105	160	119	133	97	133	95
110	65	80	104	117	72	87	107	117	72
94	66	75	112	116	92	100	110	125	90
93	68	76	97	124	81	95	101	121	81
122	76	91	104	138	96	110	97	145	92
136	94	108	92	128	82	97	88	120	81
118	78	91	116	144	98	113	94	131	87
106	68	81	104	144	92	109	90	132	86

	3 MIN AFTER INTUBATION				5 MIN AFTER INTUBATION				MMS
MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	
80	93	98	72	81	96	101	72	82	I
86	117	107	71	83	116	104	69	81	I
97	84	117	68	81	67	100	60	71	I
130	78	157	109	125	73	136	88	104	II
96	73	99	75	83	64	91	69	76	III
77	67	101	64	76	65	99	63	75	I
115	109	109	76	87	103	106	74	85	I
107	118	131	88	102	116	129	84	99	I
105	98	127	91	103	83	114	79	91	I
107	67	119	76	90	63	106	67	80	I
81	98	107	68	81	95	105	66	79	I
111	78	109	77	88	73	108	74	85	I
95	84	115	73	87	83	106	65	78	I
108	85	105	74	84	82	104	72	82	I
76	85	104	59	74	78	103	59	74	I
83	82	93	64	74	70	80	56	64	I
115	90	141	98	112	82	130	87	101	I
114	82	128	86	100	78	103	71	82	II
96	76	133	73	93	75	132	77	95	II
117	92	147	90	109	84	138	81	100	II
116	95	127	86	100	83	117	72	87	I
93	79	102	71	81	73	96	60	72	I
108	93	150	107	121	84	128	89	100	I
87	88	110	66	81	79	108	63	78	II
102	71	117	87	97	60	92	52	65	I
94	92	121	83	96	80	118	82	94	II
110	95	129	92	104	86	120	86	97	II
94	86	119	80	93	84	118	79	92	I
102	92	128	86	100	91	127	85	99	I
101	88	124	84	97	86	118	78	91	I

CL GRADE	LARYNGO DURN(S)	SIDE EFFECTS HYPO	BRADY	ARRHYTHM
I	13	N	N	N
I	9	N	N	N
I	13	N	N	N
I	14	N	N	N
IIA	18	N	N	N
I	12	N	N	N
I	11	N	N	N
I	9	N	N	N
I	13	N	N	N
I	13	N	N	N
I	13	N	N	N
I	9	N	N	N
I	11	N	N	N
I	14	N	N	N
I	13	N	N	N
I	10	N	N	N
I	13	N	N	N
IIA	17	N	N	N
I	12	N	N	N
IIA	16	N	N	N
I	13	N	N	N
I	14	N	N	N
I	14	N	N	N
I	16	N	N	N
I	15	N	N	N
I	13	N	N	N
I	14	N	N	N
I	17	N	N	N
I	12	N	N	N
I	16	N	N	N

ESMOLOL GROUP

NAME	AGE	SEX	WEIGHT	IP NO	BASELINE		
					HR	SBP	DBP
VIMALKUMAR	21	M	62	61125	78	130	80
LAKSHMIKANT	23	M	53	65306	86	120	82
AMMU	27	F	60	62668	88	112	80
PARIMALA	31	F	68	62557	68	120	78
RAMANI	21	M	55	63994	98	130	84
INBASEKARAN	31	M	65	58498	84	124	70
MYTHILI	20	F	46	62219	82	122	74
RAJESWARI	25	F	48	60820	86	126	80
GEETHA	22	F	46	62875	104	118	76
MUSINAPARVIN	28	F	62	62223	76	102	70
PARVIN BANU	22	F	43	63495	90	120	80
CHINAPONNU	35	F	50	64993	74	130	90
BALACHANDRAN	28	M	58	65027	76	126	84
TAMILMOZHI	18	F	37	65691	89	100	70
SELVI	38	F	62	66320	90	120	76
KALAIVANI	22	F	38	64673	86	116	88
NAVINKUMAR	15	M	35	63457	88	130	84
UMA	18	F	37	60214	108	122	78
MOHANAPRIYA	16	F	37	65785	90	124	84
GUNASEKAR	19	M	54	69503	92	134	80
NABISHA	27	F	52	70607	102	128	86
ELAVARASAN	26	M	66	71868	86	144	100
JAYAKUMAR	60	M	70	73577	76	132	80
RAJATHI	30	F	45	73479	78	124	78
AJITKUMAR	16	M	44	74918	80	128	74
RAMESHKUMAR	28	M	65	76471	78	116	72
NAGAMMAL	38	F	62	73917	86	126	80
RAJKUMAR	40	M	64	74570	88	124	82
KAVIARASAN	19	M	45	72153	88	126	74
SUSEELA	32	F	59	72063	88	120	86

AFTER PREMED					AFTER TEST DRUG				
MAP	HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	
97	70	117	67	77	69	101	54	66	
95	82	118	82	94	71	96	65	75	
91	90	113	78	90	93	90	69	76	
92	73	124	71	89	58	85	48	60	
99	94	124	83	97	71	91	54	66	
89	72	126	75	92	64	99	53	68	
90	81	113	74	84	67	103	57	71	
96	98	124	85	98	83	108	77	87	
87	106	111	63	74	92	105	61	73	
80	80	96	68	77	67	85	51	62	
93	83	112	75	87	73	96	62	73	
103	72	128	83	98	74	108	78	88	
98	71	125	81	96	69	100	59	73	
80	118	119	79	92	109	117	82	94	
91	86	118	74	89	67	103	57	72	
97	90	119	86	97	84	107	70	82	
99	124	131	81	98	107	88	58	68	
93	96	120	78	92	89	103	69	80	
97	104	120	77	91	91	111	69	83	
98	100	142	72	95	75	110	53	72	
100	100	121	77	92	99	98	63	75	
115	67	143	81	102	67	125	60	82	
97	75	126	77	93	58	90	60	70	
93	90	126	83	97	71	93	62	72	
92	78	124	70	88	84	112	64	80	
87	74	110	66	81	80	104	62	76	
95	74	120	78	92	71	93	57	69	
96	80	120	78	92	72	98	68	78	
91	84	114	72	86	74	94	63	73	
91	87	116	76	89	82	90	52	65	

AFTER INDUCTION				IMM AFTER INTUBATION				1`MIN AFT
HR	SBP	DBP	MAP	HR	SBP	DBP	MAP	HR
70	120	65	83	83	116	66	76	80
73	93	67	76	88	108	77	87	84
104	94	71	79	105	98	71	80	104
71	98	57	71	65	112	61	78	63
71	111	71	84	78	105	69	81	84
66	114	71	85	68	103	63	76	63
77	110	57	75	80	107	66	81	78
102	100	75	83	103	120	87	98	96
103	102	61	72	108	112	77	87	106
75	92	57	69	87	110	78	89	86
67	115	80	92	84	114	72	86	84
73	120	78	92	80	109	77	88	76
62	113	66	82	76	114	72	86	76
110	117	80	92	114	103	67	79	112
77	110	57	75	80	107	66	80	78
90	113	75	88	96	115	74	87	88
118	122	78	93	114	112	68	83	110
92	102	66	78	104	118	84	95	100
98	119	74	89	92	111	69	83	92
84	123	58	80	97	134	73	93	96
98	111	76	89	110	125	85	98	108
64	99	54	69	99	136	98	111	83
62	94	62	73	70	111	74	86	65
74	106	73	84	76	114	79	91	73
88	113	69	84	92	122	75	91	88
81	109	66	80	94	111	66	81	92
76	107	70	82	91	116	79	91	90
76	99	69	79	78	127	84	98	74
79	107	73	84	106	118	81	93	98
85	98	53	68	102	125	84	98	91

ER INTUBATION				3 MIN AFTER INTUBATION				5 MIN AFTER INTUBATI	
SBP	DBP	MAP	HR	SBP	DBP	MAP	HR	SBP	
106	53	67	78	94	50	65	76	95	
108	77	87	80	105	71	82	73	102	
96	70	79	103	93	69	77	102	80	
89	41	57	60	90	42	58	57	93	
105	69	81	77	92	62	72	72	92	
106	55	72	64	98	53	64	62	99	
104	53	66	75	101	50	66	74	104	
100	74	83	91	95	70	78	74	96	
104	77	85	102	95	48	61	102	90	
104	73	83	85	97	71	80	83	87	
97	57	69	82	92	54	67	80	94	
98	60	73	75	86	56	66	74	85	
110	66	81	75	105	61	75	74	101	
100	65	77	110	94	59	71	110	89	
104	53	70	75	101	50	67	74	104	
108	64	79	87	103	51	68	86	98	
106	64	78	104	98	63	75	102	97	
114	81	92	102	112	79	90	98	108	
105	69	81	92	106	58	74	90	107	
133	71	92	94	130	56	81	92	125	
112	78	89	98	111	78	89	92	110	
133	83	100	76	118	73	88	67	107	
109	73	85	64	107	73	84	64	106	
111	75	87	72	109	72	84	71	105	
115	65	82	84	111	60	77	78	107	
108	64	79	88	108	66	80	90	107	
104	74	84	84	100	70	80	82	98	
119	81	94	72	118	80	93	69	116	
107	66	80	90	103	65	78	88	105	
113	75	88	90	106	69	81	92	99	

ION		MMS	CL GRADE	LARYNOSCOPY	SIDE EFFECTS		
DBP	MAP			DURN (secs)	HYPOTENSION	BRADY	ARRHYTH
50	65	I	I	16	N	N	N
68	79	I	I	11	N	N	N
56	64	I	II A	12	N	N	N
43	60	I	IIA	13	Y	y	N
60	71	I	I	11	N	N	N
52	63	I	I	10	N	N	N
43	72	I	I	10	N	N	N
72	80	I	I	11	N	N	N
42	52	I	I	9	N	N	N
59	68	I	I	13	N	N	N
49	64	I	I	10	N	N	N
56	66	I	I	13	N	N	N
57	72	I	IIA	12	N	N	N
56	67	I	I	12	N	N	N
53	70	I	IIA	12	N	N	N
53	68	I	I	14	N	N	N
62	74	I	I	11	N	N	N
75	86	I	I	11	N	N	N
58	74	I	I	11	N	N	N
52	76	I	I	13	N	N	N
73	85	II	IIB	16	N	N	N
64	78	II	IIA	18	N	N	N
72	83	II	IIB	16	N	N	N
71	82	II	I	14	N	N	N
59	75	I	I	12	N	N	N
65	79	I	I	14	N	N	N
68	78	II	I	16	N	N	N
78	91	II	IIA	19	N	N	N
72	83	I	I	12	N	N	N
62	74	I	I	14	N	N	N